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Volume XXV Toronto, January, 1948 Number 4

The University of Toronto Medical Journal

(AN UNDERGRADUATE PUBLICATION)

This journal is published six times a year, November to April, by the Medical Society of the University of Toronto, to serve as a medium for student expression and to bring to the student recent articles of scientific interest.

$1.50 per volume. 25c. per single copy.

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EDITORIAL

THE CANADIAN ASSOCIATION OF MEDICAL STUDENTS AND INTERNS

Two years ago this column was occupied by a dispute between our predecessor of that year, and the Chairman of CAMSI.

The Editor felt—and at that time he had considerable justification—that CAMSI should be abandoned by this university because it served no useful purpose, and because its radical views and often unethical conduct brought discredit on all medical students.

The Chairman replied that it was only indifference and ignorance on the part of the Editor and the rest of the medical students which prevented them from appreciating the role of CAMSI. He reminded his readers that CAMSI was responsible for the army pay received at that time by the senior students, and for the arrangement by which University final exams and Canadian Council exams are written as one paper. He admitted—for he had to—that some proposals had been absurd and radical. But he assured us that more moderate policies would be followed.

The effect of this controversy was to awaken interest in CAMSI at that time. A well attended general meeting was held to discuss a brief on health insurance which CAMSI had prepared. And, most important, students whose ability gave them the respect of their fellows were elected to CAMSI offices.

After that, CAMSI was forgotten again.

It is probable that at this moment there are many students at this university who do not known that they are members of CAMSI. There are probably many more whose knowledge consists of two memories. One, being asked, and consenting with some diffidence, to pay one dollar per year as a CAMSI fee. The other, being asked to fill out a questionaire which asked innumerable questions about their medical intentions, and their finances. Because of these two experiences, many feel that CAMSI is an expensive nuisance.

We have been privileged—or rather have availed ourselves of our right— to read the minutes of two CAMSI conferences, and to discuss them with the delegates from Toronto. We have been convinced that the leaders of CAMSI have been quietly and conscientiously working to place our society on a firm basis, and to make it perform a real function in the service of Medical Students and Interns.

Firstly, by affiliation with the Canadian Medical Association, CAMSI has been freed from the suspicion that it is a politically-minded pressure group, agitating for benefits for its members irregardless of the needs and rights of the rest of society. However, because the affiliation is loose, CAMSI does not lose any of its character as an association of those being educated. It has gained the wise council, real interest, and material aid of the very capable leaders of the C.M.A. As members of CAMSI become members of the C.M.A. they do not move to a different and perhaps hostile camp, but instead remain what they have always been—a part of the organized and regular medical profession.

Then the executive has been busying itself with interests common to us all. It is axiomatic that we are all concerned with the conditions of the study and practice of medicine. CAMSI therefore began to gather information on this subject. That was the purpose of the questionaire which was distributed last year. The principle of remuneration has been very widely approved, and CAMSI has passed on its figures and recommendations to a committee of the C.M.A. which is studying the problem. The executive feels that all has been done which can properly be undertaken by a student organization, and that the problem now lies with those who would have to find the funds and administer the program.

While affiliation with the C.M.A. was advantageous the executive rightly understood that if CAMSI became connected with any non-medical politically-minded body, the students in medicine would withdraw their support, and all that had been accomplished would be lost. It was laid down therefore, as policy, that no such affiliation should ever be made. In this way our Association is protected from the danger that it might become a tool in the hands of those seeking power, or advantage.

Many important matters were discussed at the Conference which was held during the early part of November. Fuller reports will no doubt be published in

the CAMSI Journal. However, one matter of great importance was voted on. The Manitoba delegates informed the conference that students entering the first year of Medicine in their university had been asked to sign a pledge to the government that they would work for three years in rural Manitoba on graduation and that precedence in admission to the course was given to those students who signed this undertaking. No one will doubt the need of rural areas for medical help. However, the means which the Provincial Government of Manitoba has taken to supply doctors amount to coercion. No doubt that government felt that because the Medical School is subsidized by the province, they had a right to direct in some degree the way in which the product of the school should be used. However, the Government of Manitoba forgot that the only good service is willing service. It chose to ignore the right of the individual to choose his life's work. We therefore believe that the National Conference of CAMSI was right in passing a vote of censor on the Government of Manitoba for this act, and we hope that the National Executive of CAMSI will consult with the C.M.A. in order that this strange situation may be put to right.

It was a great honour to this University that the next national headquarters of CAMSI is to be in Toronto, and that the National Executive will consist of Toronto students. It is unfortunate that those students could not be elected. That they could not is a reflection more on the lack of interest of the student body, than on the democratic policies of CAMSI. In fact, the local Committee was hard put to find students willing to stand for office in the National Executive. It is to be remembered, however, that we must still elect our local CAMSI group, and that it is the Chairman of this group who will sit on the executive of the Medical Society next year.

It is our opinion, then, that CAMSI is a worth while organization, which has done much good in the past, and deserved our support. Why is it then, that it has been so inconspicuous on this Campus? It is because so many of its services are already performed for us, either by the faculty or by the Medical Society. Other universities are not so fortunate, and our support of CAMSI means much to them. But, even if it were not our duty to support and help our fellow students in other places, CAMSI would still be worth to each of us, not only one dollar, but also considerable thought. For CAMSI is looking out for our interests nationally, and we cannot do this for ourselves.

VAGOTOMY

E. B. TOVEE, M.D.

During the past four years a new procedure in the surgical treatment of peptic ulcer, bilateral gastric vagotomy, has been introduced by Dr. Lester Dragstedt of the University of Chicago. Since its inception, widespread interest has been shown all over the United States and Canada by members of the medical profession. Slowly but surely, reports of a significant series of cases are filtering through the literature; the majority in support of the procedure, the minority against it. The entire problem is far too vast to be adequately covered in this short article and the reader is referred to current surgical journals for articles dealing with various more specialized aspects.

Criticism has been levelled at the operation since its introduction by some who state that the operation is not new; that it was performed many years ago by Schiassi, Stierlin, Latarjet and others, and that, because of poor results it was abandoned. Careful investigation of all available literature would suggest that this criticism is unjustified. In none of the reports was a complete bilateral gastric vagotomy, as such, performed and we have evidence to suggest that to be assured of the best results the operation must be complete.

The theory of ulcer genesis upon which the procedure is founded suggests that ulcers occur because of a hypersecretion of gastric juice both during the day and night. During the day its digestant action on living tissues is largely destroyed because of neutralization and dilution by food, fluid, saliva and regurgitated bile and duodenal content. The important factor then, is the night secretion—a secretion of large volume and high free acidity. During the night very little of this neutralizing process goes on and, as we shall see, the gastric or duodenal mucosa falls easy prey to the destructive ingredients of the gastric juice. In a large series of duodenal ulcer patients the average volume output and free acidity of the gastric secretions were found to be roughly twice that of a similar series of normals. This would certainly tend to support the hypersecretion theory.

Similarly, much of the present experimental investigation would fit in with this conception of ulcer production. If the entire stomach of the dog is converted into a isolated pouch, the dog can be kept alive by means of a duodeno-oesophageal anastamosis. The stomach is then converted into a pilot plant for the production of pure gastric juice and the juice is led to the exterior by a mental cannula. All secretions are collected in a detachable rubber bladder. Pure juice collected by this method varies in volume from 300-1500 cc. in a twenty-four hour period. As long as the dog is kept in good nutritional state it keeps putting out the juice with little upset in its biochemical balance. If on the other hand, we withhold intravenous fluids and electrolytes the dog dies from a profound alkalosis.

Any studies on the dog must be carried out with great celerity for it has been found that if the dog is left to his own devices, without fail it will develop a typical acute punched out peptic ulcer which will rapidly go on to perforation, haemor-

rhage, or both, and the dog will die. Many have perforated in from one to two weeks and all will come to the same end if left for a long enough period of time. The ulcer is the exact counterpart of that seen in the human and it occurs in all parts of the pouch.

Here then is a dog which, before operation, was in a normal state of health with a normal gastrointestinal tract in which the gastric mucosa was exposed to *normal gastric content.* No mucosal digestion occurred. When, however, a total gastric pouch was constructed in the same dog and the normal mucosa exposed to *pure gastric secretion* a peptic ulcer developed and led to the death of the dog. This seems to be a perfect simile for the conditions of our hypersecretion theory of ulcer production in humans. During the day the human mucosa is exposed to gastric content but at night it is exposed to almost pure gastric secretion, unmixed with ingested food and only partially neutralized by regurgitated duodenal content —in other words, the perfect setup for ulcer genesis.

If we complete our studies on the pouch dog before an ulcer develops and then do a bilateral thoracic vagotomy an entirely different picture is presented. The volume of gastric juice is reduced to between a third and a half of its previous volume and the acidity shows a similar drop. After vagotomy the dog remains in good health for an indefinite period of time. A much more important fact is however, that whereas all pouch dogs prior to vagotomy develop ulcers, very few post-vagotomy preparations do. For this and other reasons Dragstedt postulated that gastric vagotomy, by decreasing the volume and acidity of the secretions, might be an effective method for curing peptic ulcers.

The anatomy and physiology of the vagus nerves become important, therefore, in the surgical approach to the whole problem. The stomach is supplied both by the sympathetic and parasympathetic nervous systems. The latter supply comes through the right and left vagus nerves which course along the sides of the lower osophagus on their way to the lesser curvature of the stomach. From here branches pierce the serosa to innervate muscle and glands. Ramifications of the vagi supply small bowel, large bowel to mid-transverse colon, and various other intra-abdominal viscera. The vagus nerves are responsible for the tonus and motility of the stomach and the nervous phase of gastric secretion. Once the vagi are sectioned the nervous phase is greatly reduced and in addition, the tonus and motility show a marked inhibitory effect. While the former secretory phase rarely returns to normal in humans, the peripheral motor mechanism readjusts itself and tonus and motility often return to a normal or almost normal state after vagotomy.

As proposed by Dragstedt the indications for vagotomy are essentially the same as those for partial gastrectomy in the same disease. In the First Surgical Division of the Toronto General Hospital the indications are a bit at variance with those of the Chicago group.

The surgical procedures previously available in the management of stomal ulcers following gastroenterostomy or partial gastrectomy have, on the whole left much to be desired. So far, the results from vagotomy in these complicated ulcers have proved in most hands to be more satisfactory than the radical surgical

measures formerly indicated. Therefore it is felt that vagotomy is the operation of choice in dealing with stomal ulcers.

As far as duodenal ulcers are concerned the treatment primarily is still a medical problem. If medical therapy, after an adequate trial, fails, the largest number should, in all probability, be subjected to partial gastrectomy. In the remainder, vagotomy can be utilized. In the duodenal ulcer group then at present, we restrict its use to the young duodenal ulcer patient between 20 and 40 years of age and those who would for other reasons—such as cardio-renal disease—tolerate the more major procedure poorly.

There is no doubt however that the whole gamut of ulcers in the age groups above forty—penetrating, bleeding and stenosing ulcers, have in the Chicago series been shown to heal. If the results from vagotomy alone have proved so good, why do we choose to do the more radical procedure as a rule? We are a bit loathe to cast overboard, immediately an operation which in the hands of the staff has proved to be so satisfactory in the treatment of ulcer. We would like to follow our present vagotomy cases for a longer perid of time before we swing over to the other extreme.

Because of the rather grave significance of ulcerative lesions of the stomach in general, even the most enthusiastic vagotomist could not quibble with the continued use of a proven therapeutic measure—a measure which effectively removes the ulcer and gives the greatest chance for permanent cure. In some 17-20% of ulcerative lesions of the stomach there is some question of malignancy. Since gastric resection for benign gastric ulcer is singularly immune to the hazards of recurrent ulceration we feel that gastrectomy is the operation of choice for gastric ulcers.

In the preoperative phase of investigation of an ulcer patient the 12 hour night gastric secretion is most important. A duodenal tube is introduced into the most dependent portion of the stomach under fluoroscopy and continuous aspiration by Wangensteen suction is carried out for the entire period. The volume and acidity are noted. On the average a normal night secretion varies from 600-800 cc. with a free acid of 20-50 units. That of the average duodenal ulcer patient is 1,000 cc. with a free acid of over 50. The insulin test—which is only a test of the integrity of the vagus nerves and of no aid to the diagnosis of ulcer—is necessary only postoperatively to assure one that nerve section is complete.

The operation may be carried out through either the transabdominal or transthoracic approach. Although on the surface, the transthoracic approach may appear to be more simple and efficient, the fact still remains that just as many incomplete vagotomies result from that approach as from the abdominal. The abdominal approach is relatively simple and gives the added comfort that the offending lesion can be visualized and any other necessary concomitant procedure, such as gastroenterostomy can be carried out at the same time. However, in the last analysis, it is only a matter of personal choice. No matter what route is used both vagus nerves and all their branches to the stomach must be sectioned to be assured of the best result.

The main features of the postoperative care are the use of continuous intragastric suction for at least 72 hours plus early ambulation. As has been stated before, the removal of the vagus innervation alone leaves the sympathetics unopposed and there is marked inhibition of both tonus and motility of the stomach. In order to prevent further overdistension of the stomach continuous suction by means of an indwelling duodenal tube is utilized. Before the patient leaves hospital the 12 hour night secretions studies are repeated and an insulin test is performed. The night secretions should have fallen to a half or a third of their preoperative level and the insulin test should be negative.

The insulin test is essentially a test of the integrity of the vagus nerves. The hypoglycemia induced by the administration of 20 units of regular insulin intravenously normally causes a marked augmentation in the flow of gastric juice. A negative test results from complete interruption of the vagus nerves and is indicated by a failure of the stimulation of gastric secretion after hypoglycemia is produced.

After discharge from hospital the patient is placed on a regular diet with few restrictions. He is warned against overeating and overindulgence. He is allowed to smoke and drink in moderation. There are still some however, with no signs or symptoms of ulcer, who do not tolerate with perfect ease certain articles of diet which had given trouble prior to vagotomy.

The important consideration as far as both patient and surgeon are concerned is the result of any procedure. In a large series of cases reported there has been a recurrence rate of roughly 2.6%. The mortality rate for the series has been 0.3%. The remainder of the ulcers have apparently healed according to clinical observation. Incomplete vagotomies have occurred with equal frequency in both the abdominal and chest approaches.

Were we to stop here I am sure that you would agree that vagotomy would seem to be the answer to the ulcer problem. However there are some definite drawbacks. Two symptom complexes are seen with moderate frequency post-operatively. They are thought to be due, indirectly, to the removal of the vagus innervation to stomach and bowel.

The first—symptoms of gastric stasis—is characterized by a feeling of fulness after meals, upper abdominal distension, severe gas belching and in severe cases nausea and vomiting. This may occur as a result of pylorospasm, hypomotility of the stomach or scar stenosis of the pylorus following healing of the ulcer. In some cases it is severe enough to necessitate the use of a subsequent gastroenterostomy.

The other postvagotomy symptom of note is diarrhoea. In some cases it is mild and temporary, in others it may be rather marked. Many forms of treatment have been tried but none has proved universally efficacious. Hydrochloric acid, paregoric, sulpha drugs and calcium carbonate have all coincided with, or caused an alleviation of symptoms in individual cases.

A few cases of acute dilitation of the stomach have been recorded. One occurred as late as six months after vagotomy and was brought on by overindulgence.

All cases were cured by gastric lavage and did not recur. In the transpleural approach a pleural effusion may occasionally be sufficiently large to require repeated aspiration. The majority do not prove troublesome. Chest pain has proved troublesome in a fair number of cases.

It is easy to see by perusal of surgical literature that the status of vagotomy varies considerably with different surgical centres throughout the United States and Canada. We believe that the operation has a definite place in the treatment of peptic ulcer. However, until we are better able to assess the long term results from the procedure we limit its use to the treatment of stomal ulcers and duodenal ulcers in selected cases, never in cases of gastric ulcer. The final answer to the problem will no doubt be supplied in the years to come, after a careful follow-up of the large series which have already been performed by various members of the profession.

CARCINOMA OF THE PROSTATE

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EDMUND R. YENDT, 4T8

One of the most vital problems concerning the urological surgeon today is the early recognition and treatment of carcinoma of the prostate gland. The magnitude of the problem was not recognized until 1900 for the disease was once thought to be a rare condition.

INCIDENCE: As a site of cancer in the male, the prostate ranks third, being surpassed only by the skin and gastro-intestinal tract. Hugh Young estimated that 20% of men over the age of 60 have prostatic disease and of these cases 20% have cancer.

The disease is rare under the age of 50 and occurs with the greatest frequency between the ages of 60 and 70. The youngest patient reported in the literature was a youth of 15.

Ravich has published an interesting paper reporting a much lower incidence of the disease in Jewish patients.

ETIOLOGY: The cause of the disease is not known. It is often associated with benign prostatic enlargement but there is no proof of an etiological relationship between these two conditions.

PATHOLOGY: Prostatic carcinoma begins in the posterior lobe in over 75% of cases. Young states that 50% of the cases seen in his clinic were associated with benign hypertrophy.

In the usual form, the tumour produces marked induration but in certain rare cases the growth is so cellular that it is soft and semifluid. Therefore it is not safe to depend entirely for diagnosis on the softness of the prostate to rectal touch. In the usual indurated form, the cut surface is smooth and does not bulge out convexly. When cut it imparts the same gritty sensation to the knife as is felt in breast cancer. The surface is dense and dry and small yellowish islets composed of carcinomatous cells can be seen separated from the more translucent fibrous tissue.

Microscopically two main types are seen. The first is the adenocarcinomatous type which at times closely simulates benign adenoma. The second type is the scirrhous—it is much the more common. In this type masses or strands of cells can be seen invading the surrounding tissue. It is associated with a marked fibrous reaction. The pathological diagnosis maybe very difficult and the pathologist may be in doubt as to whether a benign adenoma or carcinoma is present.

Locally, the disease is as a rule insidious and slow-growing and may remain confined to the prostate and periprostatic region for a long time. Metastatic extension of the disease is however usually early. This may occur via the lymphatics or blood stream. Skeletal metastases are common—the bones most commonly involved being the pelvis and sacrum, lumbar vertebrae, femora and dorsal vertebrae. Warren and his associates feel that the chief factor in the preponderance of pelvic and lower vertebral involvement is the involvement of the perineural lymphatics.

Perineural invasion of the prostatic capsule is one of the earliest changes no matter how small the primary tumour may be—these lymphatics can often be seen distended with cancer cells. The pain which is often present in the absence of demonstrable skeletal metasteses may be due to the involvement of these lymphatics. Blood stream metastasis may occur to the liver, lung and other organs. Metastasis to the spinal vertebrae may take place via the vertebral system of veins.

In over 90% of cases, the skeletal metastases are osteoplastic while secondary bone tumours from other primary carcinomas e.g. breast cancer are usually osteolytic. This difference in reponse of bone to invasion by malignant growth has not been explained in the literature.

PHOSPHATASES: The acid phosphatase activity of adult prostate epithelium is 100 or more times as great as that of any other tissue. This activity is low in childhood but at puberty it rises rapidly to adult levels. Although the urine of an adult male continues more than three times as much acid phosphatase as that of women, the normal readings of blood serum acid phosphatase are the same for men and women. Thus in males without prostatic cancer there appears to be no appreciable leakage of prostatic secretion into the circulation.

Not only is acid phosphatase secreted by normal prostatic tissue but it is also elaborated by carcinomatous tissue except where the latter is highly undifferentiated. This is true for the tissue at the primary site as well as for distant metastases. When the carcinomatous process invades lymph or blood channels, there is an escape of prostatic secretion, and then prostatic secretion becomes an internal as well as an external secretion. The phosphatases escaping into the blood can be measured by appropriate chemical methods. In Toronto, the method generally in use is that of King and Armstrong.

The following facts have proven to be consistent in all investigations:

(1) Normal serum contains less than 2.5 units percent. This normally present enzyme is not of prostatic origin for it is present in women and children as well as in adult males.
(2) In 85% of cases of suspected or proven metastatic prostatic carcinoma, the serum acid phosphatase levels are elevated.
(3) The elevated levels range from 3 to over 1000 units percent.
(4) In about 35% of elevated levels, the value is less than 10 units percent.
(5) When there is no evidence of bony metastasis about 90% of cases give readings below 3 units percent.
(7) Occasional patients with diseases other than prostatic carcinoma such as Paget's disease and hyperparathyroidism give slightly elevated readings—usually under 4 units percent.
(8) There is no correlation between the acid phosphatase levels and the extent, type or location of metastatic involvement.

Briefly then, serum acid phosphatase levels of over 4 units percent may with few exceptions be considered diagnostic of metastasizing prostatic cancer.

In nearly all cases of bony metasases from prostatic cancer, elevated alkaline

phosphatase readings are also obtained. Such an elevation is common to all conditions characterized by widespreacd acceleration of osteoblastic activity. The determination of the serum level of this enzyme is of no value in prostatic carcinoma.

The clinical applications of serum acid phosphatase readings may be summarized as follows:

(1) The method is incapable of detecting prostatic carcinoma which has not yet metastasized.

(2) The test is useful in diagnosing metastatic prostatic carcinoma when interpretations of X-rays are inconclusive. At times metastatic spread may be diagnosed before there is no X-ray evidence of such spread.

(3) In patients known to have metastases to the bones, lungs, brain or elsewhere with the origin of the primary tumour in doubt, it is possible with this method to determine if the primary tumour is a prostatic carcinoma—a matter which is now of more than academic interest.

(4) The test is useful in determining the treatment of choice in this disease. A definitely raised acid phosphatase level is a reliable contraindiction to radical prostatectomy because it indicates that the disease has already spread beyond the confines of the prostate gland. The findings of normal levels encourages radical surgery with the hope that the process has not yet spread beyond the prostatic capsule.

(5) As a follow-up measure after prostatectomy, the test is useful in detecting or ruling out metastatic spread of tumours recurring in the prostatic bed.

SYMPTOMS: Carcinoma of the prostate, like cancer elsewhere, is a symptomless disease in its early stages. The only hope therefore of early diagnosis, while the disease is still localized to the prostate, lies in the discovery of the disease by rountine rectal examination or accidental discovery during genito-urinary investigation.

In 75% of cases, malignancy arises in the posterior lobe. In this group the disease may be palpated rectally.

Since the disease does not arise close to the prostatic urethra, urinary symptoms are late in appearing. However these are usually the first to appear. The patient begins to notice frequency and difficulty in micturition. This may be due to the malignancy itself or to the associated adenoma. In a small number of cases there is early retention and overflow incontinence. Urinary retention brings with it the usual sequelae of stagnant urine, cystitis, pyelonephritis, pyonephrosis and death.

Haematuria: The occurrence of blood in the urine is relatively rare—found in only 4-8% of cases. This is because invasion and ulceration of the urethal mucous membrane is a rare occurrence and comes late in the disease.

Local Pain: Pain in the prostate region is fairly common. This may arise in uretha, penis, perineum, bladder, rectum or testicle. Pain in the bladder or urethra

may be due to an accompanying cystitis. Local pain is now considered due to involvement of perineural lymphatics.

Distant Pain: Pain in the feet, legs, thigh, lumbar, sacral, pubis, or gluteal regions may be the first symptom of the disease and is always very distressing to the patient. The pain may be due to bony metastases or malignant tissue pressing on nerve roots. It has been stated that bilateral sciatica in a male over 50 is almost pathognomic of prostatic carcinoma.

Tumour Mass: A bone tumour in an elderly male always includes secondary prostatic carcinoma in the differential diagnosis. X-ray is apt to be deceiving as some of these tumours give the sun-ray appearance of osteogenic sarcoma. Rectal examination and serum acid phosphatase levels aid in the diagnosis.

Rectal Symptoms: The rectum is protected by Denonvillier's fascia and is rarely involved until late in the disease. Involvement of the perirectal tissues may however give rise to difficult defaecation, constipation, or haemorrhoids.

Edema of legs and scrotum: This is usually a late event. It is due to obstruction of lymphatics by the malignant process.

Loss of sexual power is a comon complaint. *Priapism* occurs rarely.

General Symptoms: Loss of weight, strength, and appetite, secondary anemia as in cancer eleswhere form the distressing end stage of this disease.

DIAGNOSIS: This presents a problem to the general practitioner, urologist, and pathologist. The following laboratory and clinical points are useful in arriving at a diagnosis.

None of the symptoms is characteristic. General physical examination is usually of little aid in diagnosis unless palpable bony or lymph node metastases are present.

(1) *Rectal Examination* is the most important single procedure in the early discovery of the disease. The outstanding characteristic of the malignant gland is its stony hardness. Carcinoma should be considered likely if any area feels definitely harder than the rest of the gland. This area is often located just to one side of midline and may feel like a foreign body beneath the finger. The prostate is often fixed when first examined due to invasion of the periprostatic tissues.

Failure to appreciate malignancy is common when (1) the growth is of the glandular type and lacks the characteristic hardness or (2) when the growth arises in a spheroid of adenomatous tissue. On the other hand certain benign lesions may simulate a hard carcinomatous gland. Dense fibrosis is a common condition which may be confused. Prostatic calculi are also hard—however they are radio-opaque and they may crepitate. Carcinoma may co-exist. Tuberculosis or an infarct may feel identical to carcinoma.

(2) *Cystoscopic Examination* is necessary but often of little help in the diagnosis. Associated conditions such as bladder stones, diverticuli or benign hypertrophy may be present. Combined endoscopic and rectal examination is useful.

(3) *X-ray* of the skeletal system is indicated if prostatic carcinoma is suspected or diagnosed. Presence of metastases is important in treatment and prognosis.
(4) *Serological Tests:* The clinical application of these tests has already been described.
(5) *Biopsy:* In many cases the final diagnosis rests in the hands of the pathologist.

PROGNOSIS OF UNTREATED CASES: Bumpus has followed 485 untreated cases. The average duration of life from the onset of the first symptom was 31 months. With metastases at first examination, 66% were dead in 9 months.

TREATMENT: This is always a discouraging problem. There are a wide variety of methods of treating the disease.

Surgical Treatment: In 1904 Hugh Young performed the first radical perineal prostatectomy. With minor modifications, this remains the only method of curing prostatic carcinoma. The main points in the operation are (1) the prostate is approached via the perineum (2) a biopsy may then be taken for microscopic examination (3) the membranous unrethra is cut across at the base of the prostate (4) the gland is mobilized by separating it from the surrounding fascia (5) the complete prostate with capsule, seminal vesicles, ampullae of the vas and a portion of the trigone of the bladder are removed "en masse" (6) the urethra and bladder are anastomosed over a Foley catheter.

For surgical removal to be hopeful of success, the following clinical criteria must be met. (1) The neoplasm, on rectal examination, must not extend beyond the capsule of the gland. The gland must be freely moveable. (2) Evidence of metastases must be eliminated by physical examination, X-ray and acid phosphatase determinations. (3) The patient must be a good surgical risk with good life expectancy. The number of cases meeting these qualifications is unfortunately very small. About 1/3 of the patients have widespread metastases at the first visit. The extent of the local lesion makes most of the others hopeless for this operation. Young found 5% of his patients suitable for this operation, but most urologists consider this figure too high.

The operative mortality in Young's series was 7%. He has operated on only 184 cases in 40 years. Less skilled operators find the primary operative mortality rate higher. With the small number of operable cases it is difficult for the average urologist to become sufficiently familiar with this operation.

One great objection to this operation is the large number of patients left with urinary incontinence—approximately 30%. Perineal fistulae form another unfortunate complication.

Consequently the procedure has not been widely popular—in some places it has met with active opposition. However it should be remembered that the radical operation offers the only hope for cure, and with earlier diagnosis and improved technique the operation might be more widely appreciated.

High grade obstruction occurs in 30% of cases of carcinoma, alone or combined with benign hypertrophy. The relief of this obstruction is very important as its result is a common cause of death in these patients. Transurethal resection is

the procedure of choice in this condition. It carries a low operative mortality even when performed on the aged.

Radiation: Radiotherapy plays little part in the treatment of this disease. Prostatic cancer is for the most part radio-resistant. Some clinics however have used external radiation plus interstitial radium in treating the disease.

Hormone Therapy: This, the most recent advance in treating prostatic carcinoma, is largely due to Huggins and his associates. Huggins noted that castration or oestrogen therapy caused atrophy of adult prostatic epithelium. In 1941, Huggins selected several patients with advanced carcinoma of the prostate showing raised blood acid phosphatase levels. After castration or oestrogen administration was carried out there was a drop in the acid phosphatase levels and also marked clinical improvement. Relief of pain often occurred in 48 hours or less. This was accompanied by a return of appetite and sense of well-being. The secondary anemia disappeared and the local lesion decreased in size and hardnss.

It is interesting to note that as early as 1880, Bland-Sutton advised castration for prostatic cancer. However the procedure did not become popular and was soon abandoned.

Huggins divides the treated cases into three groups—(a) 5% of patients derive little or no benefit from either oestrogens or castration (b) 40-50% have a pronounced clinical improvement which is unsustained, the patients becoming resistant to therapy and dying within 18 months—usually with widespread metastases (c). The remainder show a pronounced and sustained improvement. Although some of the original cases are still alive, it is almost certain that all will eventually succumb to their disease.

Failure has been attributed to metaplasia of the malignant tissue to a more undifferentiated type. It was also felt that there might be an extra-gonadal source of androgen which increases after oestrogen therapy or castration. For this reason, Huggins performed adrenalectomy on three resistant cases. All the patients died shortly after the operation.

The method of control of carcinoma of the prostate varies in different centres. Huggins has maintained that castration is the procedure of choice. There is a growing popularity of the plan used here in administration of oestrogens first and castration, reserved for the time of relapse. Once the growth becomes resistant to oestrogen therapy, however, there is often little improvement following castration. Oestrogen therapy is simple to administer either orally, hypodermically or using implants. The site of action of the drug is not known but it may act through depression of the gonadotropic function of the anterior pituitary. The most troublesome side-action of the oestrogen therapy is in producing hypertrophy of the breasts. The breasts may become very painful but the chief danger is that the patient may discontinue treatment because of the discomfort. Oedema of the extremities may also result from taking oestrogens.

Stilboestrol is the drug usually used in treating this disease. It is a synthetic drug that appears to be as active as the natural oestrogens and is much cheaper. The proper dose to use is controversial and 0.5 to 100 mgs. daily have been sug-

gested as being optimum. Probably 1 mgm. daily is effective in most cases, while 2 to 3 mgs. daily are usually prescribed in practice. Some use larger doses for resistant cases.

The suggested treatment for cases of carcinoma of the prostate may be summarized as follows:

(1) Selected early cases should be treated by radical perineal prostatectomy.
(2) Cases which are beyond operative removal or in which operation is otherwise inadvisable should be treated with oestrogen administration combined with transurethral resection for relief of obstruction if necessary.
(3) Cases which do not respond or become reactivated under oestrogen therapy might obtain benefit from castration.

VENOUS THROMBOSIS IN THE LOWER EXTREMITIES

A. W. CONN, 4T8

General Consideration:

About 50-60% of all adults carry thrombi in the plantar or calf veins and this fact is verified by autopsy findings. (1) Thrombsis in the legs is important because (a) pulmonary emboli arise in the leg veins in 90% of cases (2) and cause 2-3% of all deaths, and (b) a high morbidity may result from chronically enlarged and ulcerated legs. (3)

Therefore the aetiology, pathology, classification, clinical manifestations, prevention and treatment of this condition merits considerable attention.

Aetiology:

The incidence of thrombosis is higher in the age group over 40, in winter and in northern climates. The condition is equally common on medical and surgical wards.

There are three predisposing factors to be considered:

1. *Slowing of the bloodstream.* It is generally agreed that the greatest single factor favouring thrombus formation in the lower extremities is stasis following the sudden confinement to bed of a previously ambulatory person. (4)
2. *Injury of the intima.* Mechanical trauma such as tearing and stretching operates in many cases. Infections, sprains, fractures and allergens may lead to thrombosis at the site of injury. It has been suggested that the venous thrombosis upon confinement to bed is due to inimal injury caused by the collapsed veins.
3. *Increased coagulability of the blood.* There are many states in which the blood clots more readily; severe trauma due to operation, severe plasma loss, dehydration, polycythaemia and those with carcinoma. Injury or damage to tissue produces changes in the plasma and formed elements of the blood predisposing to thrombosis. (5)

Pathology:

Thrombosis is the formation of a solid body, the thrombus from the elements of the streaming blood. A thrombus is composed chiefly of platelets and a varying amount of fibrin upon which red cells and leucocytes are arrested. An irregularity of the intimal surface of a blood vessel results in deposition of platelets which form ridges at right angles to the bloodflow. Platets liberate thromboplastin with the formation of fibrin threads which hang between the ridges and entangle red blood cells and leucocytes. (6) Most evidence indicates the origin of this process to be in the deep veins of the calf or foot, and that by propagation and accretion, the thrombus extends up the veins, not occluding the lumen but waving freely in the bloodstream. What is the fate of this thrombus? After its formation the thrombus contracts due to its fibre content. It may become absorbed due to softening because of poor nourishment and the activity of the leucocytes. If not absorbed then

(a) organization may occur with subsequent calcification, or (b) some or all of the thrombus may break loose from its attachment.

Classification of thrombosis:

The mechanism, treatment and prognosis of thrombosis necessitates a division into (a) phlebothrombosis which is venous occlusion unassociated with inflammation, the clot being loosely attached to the vein wall, and (b) thrombophlebitis which is clotting associated with and dependent upon inflammation of the vein walls with the result that such clots are firmly attached. These two groups are quite distinct in typical cases, but unfortunately there is usually overlapping.

Clinical Manifestations:

1. Phlebothrombosis:
   (a) Mild swelling of the ankle.
   (b) Dilation of superficial veins of foot.
   (c) Minor discomfort upon palpation of calf muscle.
   (d) Slight rise of pulse and temperature.
   (e) Homan's sign is positive in a moderate percentage of cases. (Homan's sign: with the knee extended, dorsiflexion of the foot produces pain or discomfort in the calf. This is due to an associated reflex muscular spasm.)
   (f) The chest attracts attention first in 20% of cases. There may be slight chest pain, cough or dyspnoea. These symptoms are often confused with those of pneumonia, pleurisy, atelectasis and coronary thrombosis.
   (g) Bilateral involvement in very common.
2. Thrombophlebitis.
   (a) Pain in the leg and tenderness along the involved vein.
   (b) Marked swelling of the involved limb with either palour and coldness or bluish discolouration and increased temperature.
   (c) Fever 101-103 F. and leucocytosis.
   (d) Embolism is a rare complication.

Prophylaxis:

Prophylaxis is the logical method of approach to this problem. Conservative measues, anticoagulants and venous ligation are the three great weapons, but the prevention of this condition should not be limited to one type of therapy.

Conservative therapy:

   (a) Elevation of the foot of the bed.
   (b) Active leg exercises, such as wiggling the toes, leg movements and bicycle exercised. (1)
   (c) Early ambulation. Allowing the elderly or debilitated person to sit with legs dangling in a flaccid position is not early ambulation. They must walk!
   (d) Elastic stockings or compression bandage from toe to groin which increase the rate of bloodflow through the deep venous system.

These measures alone have reduced the incidence of thrombosis and embolism (1) (4) but not to a radical degree.(7)

Anticoagulant therapy:

(a) Dicoumarol. This drug is given orally but requires about two days to become effective. Dosage on the first day is 300 mgms., on the second day is 200 mgms. and on the third day the amount given depends on the prothrombin level which should be below 30% of normal in order to prevent clotting. Since response to the drug varies, the prothrombin time should be followed daily. Hemorrhages sufficient to cause death have occurred, but can usually be controlled by transfusion of fresh blood and by menadione bisulphite (Vitamin K.) 64 mgms. intravenously. Dicoumarol with early ambulation has greatly reduced the incidence of thrombosis and embolism, but thrombosis may progress in spite of dicoumarol treatment. (8)

(b) Heparin. This drug may be used during the period before dicoumarol becomes effective, or it may be used alone. Heparin acts within 2-3 hours and its effect wears off rapidly. Dosage of the drug should be about 250-300 mgms. daily intramuscularly in 4-5 divided doses. The clotting time should be kept at about 4-5 times normal. (8)

Pitkin's Menstruum (a gelatin-dextrose-acetic acid combination with heparin) acts for 18-24 hours, is given intramuscularly, and apparently is quite effective. Papaverine grains 1-1½ every few hours intramuscularly should be given concomitantly. Treatment should be continued until the patient is up and around for 1-2 days. If embolism has occurred, treatment should be continued for 2-3 weeks.

Unfortunately some patients deevlop thrombosis and pulmonary embolism after the drug has been discontinued.

Venous Ligation:

It has been found that a certain group are particularly liable to thrombosis. Such cases include heart failure, coronary thrombosis, prolonged infections, post-operative surgical patients, hip fractures, those with previous thrombosis and the very debilitated. These patients are sometimes best treated by prophylactic venous. ligation.

In a series of 110 consecutive cases of elderly patients with hip fractures treated by prophylactic ligation, Allen (4) found that there were no fatal emboli and only two developed phlebitis. In a similar number of elderly people with fractured hips without ligation, there were 11 fatal pulmonary emboli and 20 with phlebitis.

When venous ligation is decided upon, it should be done within 48 hours of commencing bed rest. Since bilateral involvement is so common, a bilateral ligation of the superficial femoral vein just distal to the origin of the profundus should be done. The operation, which should not require more than one hour is done under local anaesthesia with only slight risk. Careful interruption of the normal femoral vein is harmless and without complications.

Treatment of Thrombosis:

Once thrombosis has occurred, the objects of treatment are: (a) to prevent extension of the thrombus, (b) to prevent pulmonary embolism, and (c) to correct the local impairment of circulation. It should be emphasized that when a pulmonary embolus has occurred, there is a 10-20% chance of a subsequent fatal embolus. (8) Treatment of thrombosis with or without embolism is rationally accomplished by three useful methods; anticoagulant drugs, venous ligation and lumbar sympathetic block.

Anticoagulant therapy:

Anticoagulants may be given as outlined previously. Many investigators have had excellent results shown by amelioration of symptoms, great reduction in embolism and in extension of thrombosis.

Disadvantages:

(a) No effect on thrombus already preesnt.
(b) When thrombosis is present, anticoagulants should be continued for 3-4 weeks and this necessitates hospital stay with accurate laboratory control.
(c) There may be recurrence on cessation of treatment.

Venous Ligation:

If anticoagulant therapy can halt the thrombotic process while it is still confined to the calf, and if it is equally as safe as venous ligation, then ligation may be dispensed with. As yet there is no certainty as to which is the safer technique. (9)

Ligation should be done within 72 hours following the onset of thrombosis and should be performed proximal to the thrombotic segment. If the diagnosis is made sufficiently early, then interruption of the superficial femoral veins will be adequate. If the thrombus extends above this level at operation, thrombectomy and ligation should be done when possible. This tends to avoid the more drastic ligation at higher levels.

Ligation of the common femoral vein is to be avoided because of the inadequate collateral circulation. A ligation at this site frequently results in persistent oedema, and rarely engorgement of the limb with generalized shock due to trapping of an enormous amount of blood in the leg.

When extension occurs into the iliac veins, ligation of the common iliac may be necessary. This procedure requires a major operation, and is technically more difficult.

If both common iliac veins are involved, ligation of the inferior vena cava is the answer. However, this is rarely necessary and should be reserved for those patients with repeated septic emboli. (4)

Lumbar Sympathetic Block:

This procedure is principally used in thrombophlebitis but may be used in phlebothrombosis, where pain and swelling due to vasospasm are present. Femoro-iliac thrombophlebitis is the most frequent type of thrombophlebitis, and is seen typically as phlegmasia alba dolens. The involved limb is painful swollen, white and cold.

The rationale of a sympathetic block is as follows. (4) The pain in the limb is due to ischemia which is the result of a reflex arteriolar spasm from the involved venous segment. The spasm causes the pallour and coldness and by producing anoxia of the capillary endothelium and increased permeability, results in exudation and oedema. With prompt lumbar sympathetic block, the ischemia is relieved, and pallor and coldness disappear, exudation ceases and the arteriolar pulsations quickly eliminate the odoema.

The technique is not unduly difficult. It consists of the injection with a 5½ inch needle of 20 cc. of 1% procaine lateral to the vertebral bodies of Lumbar 1 to Lumbar 4. Occasionally, a second block is required.

A block will not protect from embolism, nor can it protect from oedema if the extent of the thrombosis is too great. Therefore anticoagulant drugs are very necessary and should be combined with compression bandages and early ambulation. Ligation should only be performed when suppuration is present

Summary:

1. The frequency and importance of venous thrombosis in the lower extremities is emphasized.
2. The most outstanding aetiological factor is confinement to bed.
3. Distinction is made between phlebothrombosis and thrombophlebitis clinically and therapeutically.
4. Prophylactic measures are stressed.
5. Therapy consists of conservative measures, anticoagulant drugs, venous ligation and sympathetic block. The combination of these measures should be determined by the individual case.

I would like to thank Dr. C. C. Gray for his helpful criticism.

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PATHOLOGICAL PROCESSES OCCURRING AS A RESULT OF ACUTE HEAD INJURIES

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Part II

Secondary Pathological Conditions Associated With Head Injuries

It is important to realize that the injury imparted to the brain at the moment of impact is irretrievable and subject neither to medical nor surgical treatment, progressing to necrosis or recovery depending upon the severity of the lesion. Nevertheless, as has been already pointed out, the possibility that some of the cells affected may recover completely, necessitates maintenance of the optimum conditions favouring this result and the essence of treatment comprises the early recognition of secondary pathological developments and their proper treatment. The secondary pathological developments in which we are primarily interested are those which cause increased intra-cranial pressure, thus further embarrassing the circulation to the region of damaged tissue and further reducing its chances of recovery. The chief causes of increased intra-cranial pressure following trauma are massive intra-cranial haemorrhages and diffuse cerebral oedema.

Shock

Before proceeding to a discussion of these manifestations a few words should be said about shock-like states which often accompany the initial phases of cerebral trauma. *Primary shock* in cerebral trauma is thought due to vasomotor paralysis of central origin which is an inherent part of the phenomenon of concussion and as usual tends to respond rapidly to warmth, rest and the relief of pain. *Secondary shock,* a condition of circulatory failure, is uncommon in uncomplicated head injuries although it does occur in grave disasters accompanied by gross damage to both skull and brain tissue. It is usually associated with other injuries, such as compound fractures of limb bones, and of course may be induced by cold, pain, haemorrhage and toxins which may occur with any type of traumatic lesion. The condition is easily distinguished in conscious patients, but in patients who are deeply unconscious the differentiation between shock and a failing cerebral circulation is extremely difficult, although the distinction is perhaps academic rather than practical because the treatment of the two conditions is identical and consists primarily of early blood transfusion with oxygen therapy and the protection of the patient from any further disturbing influences such as pain from imperfect immobilization of a fractured bone. Particularly in cases where this shock-like state is a result of the head injury *per se* with a failing cerebral circulation, the combination of transfusion with the inhalation of 100% oxygen by nasal tube is an invaluable adjunct to therapy for as has been already pointed out, anoxemia is the immediate factor responsible for rendering irreversible any cerebral damage

that has been produced at the moment of injury, and the combination of added haemoglobin within the blood stream and increased oxygen tension in the alveoli, is the best possible means of increasing to the highest point the oxygen carrying capacity of the blood in an attempt to obtain optimum conditions for recovery.

Massive Intracranial Haemorrhage

Space-occupying intracranial bleeding may occur at any level from a zone immediately subjacent to the skull to within the ventricle and in order from the surface this bleeding may result in the formation of extra-dural or sub-dural haematomata, profuse subarachnoid bleeding, intracerebral haemorrhages or bleeding into the lumen of the ventricular system. The production of a massive haemorrhage sufficient in degree to cause increased intracranial pressure usually requires the rupture of a large vessel which may be arterial but is more commonly venous in type. Popular attention has long been focused on the middle meningeal vessels because of the typical picture often produced as a result of extradural haemorrhage from this source causing the arresting syndrome of concussion and recovery followed by subsequent loss of consciousness. These vessels are most commonly injured as they run in a bony groove to which the dura is firmly attached at the region of the pterion and the diagnosis is often suggested by radiological evidence of a fracture line crossing the grooves formed by the vessels on the inner surface of the skull. The formation of a space-occupying haemorrhage in this region depends primarily upon the degree of adherence between the dura and the skull, and in keeping with the fact that the dura can be stripped from the endocranium with increasing difficulty as age progresses, these lesions are most common in the younger age group. The dura in addition is more easily separated from the overlying skull in cases where the initial trauma has been severe, particularly if it is associated with a gross fracturing of the temporal and fronto-parietal regions which has already loosened its attachment considerably, so that one must realize that secondary extradural haemorrhages may also develop in association with *severe* primary head injuries and must be considered where there is a regression in the level of consciousness without an intervening period of recovery or improvement.

Other types of massive intracerebral haemorrhage are usually venous in origin, and by no means uncommon, affecting particularly the communicating veins which drain cortical veins into the large venous sinuses and the rupture of which is associated most commonly with profuse subdural and subarachnoid bleeding. These fragile vessels are usually involved at the vertex where they run forward in contact with the under surface of the dura and firmly attached to it, and then have a short straight course across the subdural and subarachnoid space to enter the superior longitudinal sinus and may be easily torn when the cortex is displaced, as occurs in the acceleration types of head injuries.

Extradural Haematoma

This complication, occurring as described above, represents less than 1% of the pathological lesions developing in any large series of head injuries but warrants

attention because it represents a condition which will be rapidly fatal if it passes unrecognized and which properly treated, should result in complete organic recovery provided there is no associated brain injury. Although commonly occurring as a result of haemorrhage from middle meningeal vessels, it is occasionally associated with diploic bleeding or bleeding from dural venous sinuses. Bleeding from diploic channels is probably the most common cause of extradural haemorrhage but it is rarely of clinical significance because the layer of blood remains thin and does not commonly produce a space occupying lesion. When it is profuse it arises from numerous widespread areas and is associated with gross fracturing of the skull. Sinus bleeding, on the other hand, is fortunately uncommon because, when it does occur, the bleeding is profuse, despite the low venous pressure, and causes rapid progression of clinical signs. The rarity of this haemorrhage is explained by the fact that these large dural channels are not embedded in deep bony grooves, except for the sigmoid sinuses, and are not necessarily lacerated when the overlying bone is fractured, but when they are damaged, the sinus walls, being fibrous and rigid, do not collapse or contract as happens when arteries are damaged and the bleeding is often grave indeed. Rowbotham (1) rightfully emphasizes the fact that any obstruction to the venous return to the heart will greatly aggravate these venous haemorrhages within the skull and this is particularly true in laboured respiration or where there is any compression of veins in the neck, so that the importance of maintaining a clear air-way and a suitable position of the head and neck becomes apparent and cannot be overemphasized

Subdural Haematoma

Acute subdural haematoma occurs most commonly as a result of lacerations of communicating veins joining the superior longitudinal sinus but the blood tends to trickle downwards to the most dependent part of the subdural space and therefore the greatest bulk of the haemorrhage is found over the outer surface of the temporal lobes. If the bleeding vessel becomes sealed over before the haemorrhage has reached the size of a space-occupying lesion, symptoms may not result and the healing process takes place by the ordinary methods of resolution. However, in a definite proportion of cases, the haematoma becomes enveloped in a fibrinous membrane which is later organized by mesothelial invasion, the inner portion of the membrane tending to remain thin and non-adherent to the arachnoid but the outer wall thickening and becoming adherent to the dura, so that it is difficult to separate these two membranes without the rupturing of small blood vessels which have grown from the dura into the lining of the clot. This chronic subdural haematoma may at some stage in its life history cause symptoms, the symptoms resulting from the fact that the encapsulated clot begins to swell, causing local pressure effects and increased intracranial pressure. Several theories have been advanced to explain this late increase in size, and as it is common to find fresh blood within the clot at operation, Putnam (16) has suggested that oozings from newly formed vessels accounted for the increase in size. However, the most popular conception at the present is based on the fact that Gardner (17) has proven the membrane of

the haematoma to possess semi-permeable properties and therefore as the clot disintegrates its molecular concentration increases with a resultant rise in osmotic pressure, and the increase in size is dependent on the osmotic interchange from the surrounding subdural space.

Subarachnoid Bleeding

Rowbotham (1) believes that this is the most important and most common form of massive intracranial haemorrhage after trauma, with its appearance in over 75% of patients who have been unconscious more than an hour after injury. Symonds (18) reports that typically in these cases the cerebrospinal fluid taken at the time of the initial lumbar puncture shows: 1. an even admixture of blood in all specimens; 2. absence of coagulation and 3. pink, brown or yellow discoloration of the clear supernatant fluid when the red blood cells have settled. It is surprising how rapidly this extravasated blood is haemolized and its products removed by means of the arachnoid villi, and it is not uncommon to find no frank evidence of haemorrhage within a few days of injury, although xanthochromic staining may persist for many weeks. The presence of subarachnoid blood is commonly thought the basis for the clinical evidences of meningismus often exhibited by patients who have suffered head injuries, and also as a result of the irritation of the meninges by its presence, to represent one basis for the restless state which is so common in patients with impaired levels of consciousness. This restlessness in itself is an important factor in increasing the bleeding, by promoting cerebral congestion, and therefore its control is an important feature of any therapy designed at preventing the occurrence of these secondary manifestations.

Other Types of Massive Intracranial Haemorrhage

In acute head injuries, *intracerebral haemorrhage* may occur near the surface as the result of superficial laceration, particularly in contre-coup injuries, or be deeply situated, resulting from the rupture of cerebral vessels which have been the seat of pre-existing vascular disease. Fortunately this latter type is extremely uncommon, but must be remembered in old people who develop evidences of secondary space-occupying haemorrhage when no extradural or subdural haematoma are found at the time of exploratory craniotomy.

Intraventicular haemorrhages are thought to result from either tearing of the choroid plexuses or from rupture of a large intracerebral haemorrhage through the ventricular wall. If profuse, they are invariably fatal and most commonly occur in children. The recognition of these conditions in time to effect adequate surgical treatment is difficult and the outlook poor.

Diffuse Cerebral Oedema

Probably more conflicting opinions have arisen with regard to the presence or absence of space-occupying cerebral oedema than any other complication of head injury, but with the publication of recent evidence it seems to be apparent that the importance of oedema has been over-emphasized in the past. Shapiro and Jackson (19) point out that this diagnosis should be reserved for cases dis-

playing an increase of fluid in the paravascular and peri-cellular spaces, and, after measuring the water content of traumatized brains, they felt that, although the brain was frequently enlarged after head injuries, the enlargement was caused by ventricular dilatation and blood vessel engorgement rather than actual oedema. Greenfield (20) was unable to demonstrate a single case of generalized oedema in his study of traumatised brains. Undoubtedly a localized form of oedema occurs around areas of contusion but Jefferson (21) and others believe that oedema of severe grade is limited to the immediate periphery of these focal areas and that a generalized form exhibiting the typical areolar honey-combed appearance due to distension of these peri-cellular spaces is extremely rare.

Nevertheless, it does appear that increased intracranial pressure may appear in the absence of any other space-occupying lesion, and these cases are probably due to a diffuse cerebral oedema, the causation of which is difficult to explain. Linell (14) has suggested that it is due to the disruption of the attachments of astrocytes from the walls of vessels, and other authors have described its occurrence on the basis of increased capillary permeability due either to vasomotor paralysis or the accumulation of abnormal amounts of metabolites within the tissues. At any rate, as Rowbotham (1) points out, once established, this lesion causes a vicious circle, the oedema resulting in increased intracranial pressure which secondarily causes venous congestion, with further capillary stasis and subsequent increase in the oedema.

Although Russell (8) found increased intracranial pressure in thirty of forty-nine cases in which there was no evidence to suggest other causes of increased intracranial pressure, Botterell and Wilson (22) were unable to demonstrate any abnormal increase in pressure in 77 cases of minor head injury and only 4 cases in 125 patients who had been subjected to more severe injuries, exhibited pressure over 300 mm. of water. There were 21 patients in this series with a pressure varying between 200 and 300 mm. of water, but from their observations it was concluded that cerebral oedema with increased intracranial pressure rarely played a part in producing post-traumatic coma or confusion. This is in keeping with our own results, for in our experience significant elevations of intracranial pressure (over 250 mm. of water) were always associated with space-occupying intracranial haemorrhages and in 12 patients who died as a result of an acute head injury and at post mortem showed no evidence of other intracranial space-occupying lesions, the pressure as recorded by lumbar puncture was below 200 mm. of water in every case. We agree therefore, very strongly with the opinion that cases showing elevation of intracranial pressure must have all other types of space-occupying lesions ruled out by exploratory craniotomy before accepting a diagnosis of cerebral oedema and treating the patient by dehydration methods or by the drainage of cerebrospinal fluid in large amounts.

External and Internal Hydrocephalus

External Hydrocephalus

It has been frequently shown microscopically that the pacchionian granulations may be filled with extravasated blood in head injuries, and external hydrocephalus is thought to result from a faulty absorption of cerebrospinal fluid due to this blockage. There may be in addition a laceration of the arachnoid which allows cerebro-spinal fluid to leak into the subdural space and there accumulate, occasionally to space-occupying proportions. These hygromas as they have been called may be acute or chronic in nature. In some cases the protein content of the hygroma is much higher than that of the cerebro-spinal fluid and it has been suggested that this is due to a transudation into the subdural cavity resulting from injury to its lining cells rather than a rupture of the arachnoid membrane. Almost any surgeon who has been associated with the treatment of head injuries has had the experience of finding these collections of clear fluid deep to the dura when exploring for a possible intracranial haemorrhage, only to be pleasantly surprised that the symptoms have been relieved post-operatively by their drainage; and McConnell (23) has recently suggested that similar localised collections of fluid may be the basis of prolonged post-traumatic amnesia following head injury. He has shown that collections of fluid other than blood are occasionally found in the subdural space and that when these are drained there is a rapid sustained improvement of the patients's mental state, and on this basis thinks that local exploration in such cases is indicated.

Internal Hydrocephalus

Dilatation of the lateral ventricles is also occasionally found following head injuries and this condition may also occur in acute and delayed forms. The acute form is probably most often due to the obstruction of the Sylvian aqueduct by clotted blood or sloughed-off brain tissue, although Courville (24) suggests that it may result from increased secretion of cerebro-spinal fluid resulting from abnormal activity of choroidal and ependymal cells due to paralysis of the vaso-constrictor fibres.

The delayed form may be of two different types. In the first place it develops slowly as a result of stricture of the aqueduct by bruising of its walls, or obstruction in the roof of the fourth ventricle by organizing extravasated blood. In the second instance the dilatation, termed a hydrocephalus ex vacuo occurs as a result of some change taking place primarily in the brain tissue—either traction on the ventricular walls by a contracting cerebral scar or protrusion of the ventricle into an area of softening by the rhythmic ventricular pressure changes occurring with cerebral pulsation. O'Connell (25) has stated that the cerebro-spinal fluid pressure varies rhythmically through an average of 80 mm. every 3-4 seconds and exerts intermittent thrusts of pressure on the injured or weakened ventricular wall.

Recognition and treatment of these rare complications is a specialized neurosurgical responsibility.

"Pulping" of Frontal and Temporal Poles

During the early stages of the war it was recognized by Botterell and Stewart (26) in England that there was a form of increased intra-cranial pressure arising several days after the injury and dependent upon severe "pulping" of the frontal and temporal poles, commonly of contre-coup character. Intra-cerebral haemorrhages, of course, both superficial and deep in location and often of considerable size may be related to these areas of laceration of cortical tissue, but the lesion described by these observers depends not upon continuing haemorrhage but rather upon a massive cerebral softening which progresses to a gross localized cerebral oedema, for the production of increased intra-cranial pressure. In these cases, the damage to the brain tissue with its associated blood clot, is so great, that following the release of the breakdown products of protein degeneration in the necrotic area, the subsequent increase in colloidal osmotic pressure is sufficient to cause a localized oedema of such proportions that increased intra-cranial pressure results. Occasionally this may promote the vicious circle already mentioned that leads to a diffuse cerebral oedema as a terminal occurrence. The importance of recognizing this condition lies in the intra-dural exploration it entails in cases where negative exploration has been carried out for extra-dural or sub-dural haematomata. By the end of a week this area has become fairly well demarcated from the normal brain and may be evacuated with relative ease by suction, and the results obtained are as gratifying as those with the more dramatic extra-dural haematoma.

Meningitis

In cases with x-ray evidence of basal fracturing of the skull, one must be on guard for the occurrence of meningitis, and particularly is this true in those cases exhibiting cerebro-spinal fluid rhinorrhoea, or otorrhoea, for before the advent of sulphonamide and penicillin medication, this complication took a considerable toll of life. In the acute forms, it is associated with increased intra-cranial pressure and the resultant impairment in conscious level often suggests a secondary intra-cranial complication, such as haemorrhage or oedema, but the diagnosis may be verified by the cellular content of the spinal fluid, in which organisms are frequently to be seen at this stage, and systemic evidences of infection.

Once this complication has developed an attempt is made to control the infection by intrathecal and intramuscular penicillin therapy coupled with one of the sulfonamide drugs, but it is important to realize that this does not attack the underlying cause of the infection, and if the cerebro-spinal fluid leakage persists, surgical treatment is indicated to close the defect after the patient has recovered from the meningeal infection.

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Feature

R. K. GRAHAM, Editor

THE PRACTICE OF OBSTETRICS AND GYNAECOLOGY

H. B. VAN WYCK

Professor of Obstetrics and Gynaecology

This dual specialty would be better described by the shorter name Gynaecology, if that term could be used to include the function and pathology of the reproductive organs in both the pregnant and the non-pregnant state. Such a limitation in nomenclature would help to settle the controversy that has disputed the necessity of the union between Gynaecology and Obstetrics. In most Universities, these two are regarded as closely knit divisions of one subject; and the union in our teaching departments is the result of this viewpoint; that neither the Obstetrician nor the Gynaecologist is properly trained without the mastery of both fields. The practitioner of this specialty has a field of no mean dimensions. He begins with human life before its genesis; follows it through its prenatal development to its birth; then centres his studies on the female during puberty and the reproductive period to begin again with birth and end with the menopause. He must master a limited field of anatomy, larger fields of special physiology, biochemistry, and pathology; learn the technique of obstetrical procedures and gyneacological surgery. After graduation and the final year of rotating internship, the young graduate, if he chooses to become a gynaecologist, must secure three or four years post-graduate training—preferably one of these years being spent in one of the basic sciences.

Upon what grounds should the choice of such a specialty be made? Not certainly as would appear on superficial consideration, because of a predeliction for surgery as opposed to medicine, because the specialty overlaps both domains. The first can claim as large an interest in endocrinology as general medicine itself; the disorders of the female reproductive system are both medical and surgical. Nor can the choice be founded upon a desire for a life of pleasant medical dilettantism, nor any love of seeing the sun rise. One is, with any luck, usually in bed at least by that time. On second thoughts, I can find no adequate reason for the choice unless it be that one loves to travel by moonlight, when there is no traffic; the thoroughfares are free of dust, no disturbing temptations come from open pubs, and it may be some satisfaction to realize that by getting some major jobs done at night, these do not interfere with one's chores and routines in the hospital next morning. These delights are of course compensated for by the unpleasant though flattering tendency of humanity to plan to reproduce itself under your supervision—no one else will do—during week-ends desirable for a trip to the "cottage" or by the utterly puzzling facility with which nature can put full term to one of its anticipated fledglings after the first act of a delectable musical comedy.

Nor do I know of any other medical problem about which so much unsolicited and irresponsible advice can be given than a tedious labour. But in spite of all this and haemorrhage too, I know of no gynaecologist or obstetrician worthy of the name who would trade his profession for any other, and what seems curious they all seem to have tremendous practices. They enjoy their work so much that they seem to live for ever which is something to make pause to think anyone who hopes to succeed them. So that all in all, if a young medico makes such a choice, is willing to spend enough time in training, is willing and able to work irregularly and for long days and nights together, will forgo many an enticing plan, will be willing to miss many church sermons because of the difficulty of Sunday morning attendance—in a word will cheerfully put the capricious and often precipitate whims of his patients before the domestic delights of cleaning the snow and tending to the furnace etc. etc., such a choice will never be regretted.

On the whole, no work is happier than obstetrics, and the comparatively low mortality of gynaecology (malignancy excepted) means that the gynaecologist works in a cheerful environment. Of the gynaecologist's wife in all this turmoil and absence, all that need be said is that I know of no domestic alienations—and why should there be—the husband has specialized in women and knows how to placate the sex.

After completing training, the young gynaecologist will proceed to a fellowship degree or certification. Granting of certificates of qualification to specialists in Canada is carried out by the Royal College of Physicians and Surgeons of Canada. The Council of the College appoints a Committee to carry out the function of certification. The members of this Committee are nominated by the functioning organization of the speciality.

The examination for certification in Obstetrics and Gynaecology at present is partly written and partly clinical and oral. Two papers are set, one covering the principles and practice of Obstetrics and Gynaecology and one on the application of the basic sciences necessary to the proper understanding of the specialty. The examination is conducted jointly by two gynaecologists. The qualifications for admission to the examination are satisfactory moral and ethical standing; graduation from a medical school approved by the Council; the production of documentary evidence of supervised study or practice during an aggregate period of at least five years after graduation. One year must be spent in a general interneship approved by the Council, and two years must be spent in study and training in Obstetrics and Gynaecology in hospitals organized for that training. A period of the training is to be spent in the study of internal medicine or general surgery and the basic medical sciences. The candidate shall have spent the remaining portion of the 5-year period in the study or practice of the specialty. At least four months prior to the date of the examination, the candidate must forward to the Honorary Secretary of the College a completed application form together with the required documentary evidence, and the candidate will be admitted to examination only if the course of study and training is satisfactory to the Credentials Committee of

the College. The examinations are held from time to time as determined by the Council of the College.

To the gynaecologist endowed with the instinct and will to enquire into and explore the unknown—the urge for research—his field opens up ever receding horizons which lure him forward. The mystery of life itself as exemplified in the limited and biologically ordered symbiosis of pregnancy, or in the strangely related disordered and parasitic phenomena of malignancy, is in the very focus of his vision; and perhaps in the organic co-ordination between the invasive chorionic villus and the decidual host lies ready for discovery the secret of cancer. The curious investigator may ponder the relation between ontogeny and phylogeny in the growing ovum and attempt to unlock the teleology of evolution and its relation to determinism. With the development of the ovum, the metabolic dualism which in harmony is called normal reproduction and in dysharmony the toxaemias of pregnancy, has prodded the scientific imagination to attempt the answer. To bring further safeguards to the function of reproduction, the gynaecologist may find himself over the boundary of physiology and biochemistry invading the fields of physics, radiology, serology, and genetics; and, in the relation of reproduction to the passage from youth to old age find the answer to many a riddle. To advance knowledge in a realm thus rich in tantalizing potentialities may well attract the keenest minds.

In the history of gynaecology and obstetrics, the successes of the present and the hopes of the future must not be allowed to obliterate the memory of the strivings and victories of the past. The aspirant to a career in this specialty may well acquaint himself of the long, slow history of advance to the present standards. Here lies buried a rich lore of wisdom and folly, brilliant and daring intuitions, and toil, always laborious, often of pedestrian monotony and, at its great moments, heroic. Regard for the past is a precious faculty of civilized man. If the past be nothing but a dead hand, if it be not regarded as the matrix of the present and the future, he will fail to read its lessons or to shun the closed paths chosen only by the blind.

In the cycle to which we belong, we can see only a segment; to look ahead we must often look back many a century.

The compass guiding the advance in gynaecology has pointed in every direction. The priests of ancient peoples ascribed the diseases afflicting women to the malign influences of unpredictable and supernatural forces of the moon, the planets, and the gods themselves; and primitive gynaecologists treated their patients with prayers and amulets to propitiate their hostile deities. Today, after centuries of vacillating progress, the mysteries of hormones and vitamins occupy the stage, and the cynic may find some amusement in pointing out that the wheel has gone full circle from libations in the Temple of Appollo to psycho-analysis. To silence the scoffer, one needs only to point to the record of achievement in scientific development during the past hundred years. The upward trail as it may be surveyed in retrospect has been hallowed by such pioneers as Semmelweiss, Holmes, Simpson, MacDowell, Pasteur, Virchow, Lister, Bernard, Zondek and a host of others. Technical processes have been perfected and surgical procedures restricted

to their proper field, while in the laboratory new concepts in great profusion are bursting into view. Gynaecology and Obstetrics share a rich heritage. Whether the present period of active advance will be followed by the periods of stagnation which the centuries have often recorded is a query that the present state of our world presents. Science has always advanced wherever men are free and religions are tolerant. To-day freedom must be prized and defended as never before, and the first and most strategic of its bastions to fall would be scientific freedom, whether the foe be within or without the State. Without a disciplined freedom the practice of medicine in any of its fields becomes degraded and menial.

The standard of efficiency of the specialty of Obstetrics and Gynaecology in Canada must and will be maintained on an ever higher level. Undergraduate medical education today equips the practitioner with a grade of training undreamed of by past generations; and general practitioners who have taken adequate post-graduate training, will continue to attend perhaps the majority of the confinements in this country. Hence the specialist must by sound additional training be worthy and able to assist and advise his colleagues in the community in their more difficult problems. No mere self-acclaim will enable him to win their confidence and respect—the only road to this desirable and useful place in a community is thorough, long, and arduous training.

Of advice to the aspiring young gynaecologist, one would note that the sounder the knowledge of basic science and general medicine, the more efficient the gynaecologist becomes. A wide range of reading of the few classic and the many current contributions, discussions, travel to other centres, and intense application to the duties at hand, are means to success; but one's advice would not be complete if one did not also stress the development of the personality and character that makes a good gynaecologist a better one;—equanimity, courtesy, gentleness, and a sympathetic understanding. Roughness of crudeness in word or deed do not appear to advantage in an obstetrical environment.

To one who is fitted for this work by training and natural endowment, the specialty of Obstetrics and Gynaecology offers a career of useful service well worth the investment of all one's life and energy.

Abstracts

E. R. YENDT, Editor

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Copper Sulphate Method for the Rapid Estimation of Whole Blood Requirements. J. C. Edwards: Arch Surg. *55,* 1-12 (1947).

Edwards used the copper sulphate specific gravity method (Phillips et al.) to determine the hemoglobin and total protein concentration and the hematocrit in one hundred sixty-six patients with severe orthopedic injuries. The values so obtained were found to agree closely with similar estimations made by the more usual laboratory methods. He calculated blood requirements on the basis that 50 ml. of whole blood will, on the average, give a rise in the hemoglobin concentration of 0.9 Gms. per 100 ml. During the four day preoperative and two week postoperative periods, these patients required, on the average, 2,360 ml. of whole blood. An average of 1,470 ml. of whole blood was required by each of ninety-eight patients admitted with soft tissue wounds of the abdomen, the chest or an extremity. Upon admission to hospital, half of all the patients had hemoglobin concentrations below 12 Gms. per 100 ml., total protein concentrations below 6.6 Gms. per 100 ml. and hematocrits below 40%. Changes in these values, before and after treatment, were found to be of value in following the course of the patients' conditions. The copper sulphate specific gravity method for determining hemoglobin and total protein concentrations and the hematocrit, with the aid of a line chart, proved to be a simple yet accurate procedure for clinical use.

W. G. B. C.

Annual Report of the Committee on Therapy and the Subcommittee on Streptomycin of the American Trudeau Society on Streptomycin Therapy of Tuberculosis. J. Amer Med. Assn. *135,* 641-645 (1947).

In the opinions of the members of the committee and subcommittee, the following conclusions and recommendations appear to be justified on available evidence regarding the use of streptomycin in the therapy of tuberculous infections:

(1) Intensive parenteral and intrathecal streptomycin therapy is advised for treatment of tuberculous meningitis. Early diagnosis and prompt treatment are essential for good results, in this and all other conditions.

(2) Streptomycin therapy is advised for treatment of acute hematogenous miliary tuberculosis.

(3) Streptomycin therapy is advised for the treatment of more severe cases of tuberculous laryngitis and ulcerating tuberculous lesions of the mucous-mem-

branes of the oropharynx. Combined parenteral and topical treatment is recommended at present.

(4) Streptomycin therapy is advised for treatment of progressive ulcerating tuberculous lesions of the tracheo-bronchial tree. It will not benefit fibrous strictures.

(5) Streptomycin therapy is not indicated at this time for the treatment of all types of pulmonary tuberculosis. Most suggestive results have been noted following treatment of recent but extensive, progressing pulmonary lesions. Streptomycin cannot be recommended at the present time for the treatment of (a) chronic fibroid or fibrocaseous pulmonary tuberculosis, (b) acute destructive and apparently terminal types of pulmonary tuberculosis, or (c) minimal or early moderately advanced pulmonary tuberculosis with favorable prognosis.

(6) Streptomycin therapy is suggested for further trial as a remedy for the symptoms of acute ulcerative tuberculous enteritis.

(7) Streptomycin therapy is recommended for the treatment of tuberculous draining cutaneous sinuses. Sinuses associated with tuberculous empyema are less likely to respond to treatment.

(8) Streptomycin is not recommended for treatment of chronic empyema of tuberculous origin.

(9) More extensive observations will be necessary for the justification of the use of streptomycin in (a) prophylactic treatment before and after surgical procedures, (b) treatment of tuberculosis of the genitourinary tract, (c) treatment of tuberculosis of the bones and joints, (d) treatment of tuberculosis of the skin, (e) treatment of tuberculous lymphadenitis without sinus formation, and (f) treatment of ocular tuberculosis.

(10) The following toxic potentialities of streptomycin must be kept in mind whenever its use is considered and must be compared with the hazards of the disease to be treated: (a) Disturbance of vestibular function, (b) Deafness (very rare), (c) Serious renal damage (rarely in the absence of pre-existing renal disease), (d) Cutaneous rashes, and, rarely exfoliative dermatitis.

At present it is recommended that the total parenteral dose for 24 hours be 1-2 Gms. and that the intramuscular injections be given at intervals of 4-6 hours over a period of 3-4 months. Further studies have yet to be made to determine the minimal effective therapeutic dosage and how this dosage should be modified to give optimal therapeutic results.

The disappearance of drug sensitive strains and their replacement by drug resistant ones is a serious handicap to streptomycin therapy. Neither the conditions which encourage this change nor its frequency of occurrence is known. Therefore streptomycin treatment should be avoided when other treatments are available.

W. G. B. C.

Reducing Mortality in Gastric Carcinoma Gilson Colby Engel; J.A.M.A., *135*: No. 11, Nov. 15, 1947.

Carcinoma of the stomach kills more people annually than carcinoma of any

other organ, taking 38,000 lives each year in the United States, In various reported series, less than 20% of cases when diagnosed are operable with hope of cure.

From his series of 243 cases, the author reports:

28% not considered for operation (clinically incurable).

36% opened, and closed because of intra obdominal findings.

18% suitable for palliative surgery.

18% curative operation undertaken.

Two reasons are given for the low operative possibility-difficulty in diagnosis, and delay all along the line.

The only constant early symptoms felt to indicate further investigation were a tired and weak feeling, loss of appetite, loss of desire for meat, and mild distress related to meals.

Investigations considered important though not individually infallible were: (1) X-ray examination of the stomach (2) gastroscopy (3) fractional test meal (4) serum proteins and (5) complete blood count.

Delays occur (a) in onset of symptoms (b) in the patient's consulting a physician (c) in adequate investigations (d) in surgical consultation and operation and (e) by the patient in following the advise given.

Operation must be thorough and expertly done. The author reports mortalities of 0.2% in exploratory operations, 2.8% in subtotal gastrectomy, and 18% in total gastrectomy, in contrast to 100% mortality from the disease unoperated.

Reduction in mortality is the aim of these suggestions by the author: (1) Group screening by X-ray (2) Gastric X-rays with each annual physical check-up (3) Education of groups to see their physician for gastric symptoms of over two weeks duration, or for fatigue and loss of appetite (4) Gastric X-rays for these latter complaints (5) Additional studies as listed above to be carried out (6) Treatment for 4 weeks followed by repeat of X-rays, of any patient with a gastric lesion (7) Operation in failure of ulcer to heal radiologically after 4 week's medical treatment, even though symptoms have disappeared (8) Operation where X-rays or gastroscope shows gastric carcinoma, and (9) Extensive operation.

T. K. A.

Complete Heart Block in Younger Age Groups. J. H. Crawford, N. J. DiGregorio. American Heart Journal *34*: 540-551 (1947).

Fourteen cases of complete heart block are presented, the oldest patient being 39 years old. Two of these were of congenital origin, six were due to rheumatic fever, two to arteriosclerosis, three were probably of congenital origin, although evidence of this was inconclusive, and in one case no cause could be determined.

In block resulting from rheumatic fever, diphtheria, pyogenic infections and trauma, the mechanism of the block is fibrotic change in the bundle which prevents it from functioning properly. In syphilis the interrupting lesion is either an actual gumma or fibrous tissue. In tumour, malignant tissue interrupts the bundle fibres, while in ulcerative endocarditis, the ulcerative process severs the bundle. The commonest lesion associated with congenital complete heart block is

a patent interventricular septum. Finally, variations in the circulation to the bundle may effect its conductivity. This is the mechanism in the older age groups where coronary arteriosclerosis is a factor.

The most important factor in the maintenance of circulatory efficiency in complete heart block is the increased output per beat. Several of the cases illustrate well that the individuals with complete heart block may carry on strenuous activity, their cardiac output increasing with exercise, producing a minute volume only slightly below that of a normal individual. The increased cardiac output results from an increased inflow in the heart due to a longer diastole, resulting in:

1. Increased in the length of the ventricular fibres.
2. Increased intraventricular tension.
3. Altered surface-volume relation which influences the effectiveness of a given muscle shortening, due to the fact that the volume of sphere increases more rapidly than the surface, thus causing a greater output per unit of shortening of muscle.

Many cases in this younger age group present no symptoms at all and lead a normal life. The symptoms and signs are determined more by the presence of other abnormalities.

Where complete heart block is the main abnormality, the outlook is good, particularly in the congenital type. The prognosis depends mainly on the presence or absence of serious complicating cardiac pathology.

B. G. G.

Pleurodynia John J. Finch Jr. New England Journal of Medicine *237*: No. 17, 621-623.

An acute febrile illness called "epidemic pleurodynia" or "devil's grip, is outlined in this article, as it appeared in a recent outbreak in Boston. Three cases, chosen from over 100 admissions during the epidemic to Boston City Hospital are described to point out the diverse manifestations of the disease.

Etiology as yet is unknown. Characteristically, there is no prodrome. Onset is very sudden, with the initial symptom being pain of varying intensity in the lower thoracic or upper abdominal region, which may shift from side to side or be referred to shoulder or scapula. Paroxysmal hyperesthesia in the area of distribution of the pain usually follows.

Fever rises as high as 104 degrees F. coincident with or soon after the onset of pain, only to fall back to normal in twelve hours, after which there may be one or more recrudescences of pain and other symptoms. Between recrudescences, the patient may be entirely asymptomatic. The fever may run an irregular course up to ten to fourteen days, and once permanently remitted, there are no further symptoms except for an occasional twinge of pleurodynia.

Physical findings are as a rule limited to the fever, splinting of chest muscles, and upper abdominal or thoracic tenderness A pleural friction rub appears occasionally at some time during the course of the illness or during the convalescent period. Eosinophilia has been described in earlier epidemics.

A diversity of other symptoms and signs have been reported, leading to misdiagnosis in many cases. It has been confused with pneumonia, influenza, infectious mononucleosis, non-paralytic poliomyelitis, lymphocytic choriomeningitis gastroenteritis and acute surgical conditions in the abdomen.

The course is benign as a rule, but complications such as pericarditis, orchitis and jaundice have been reported.

H. M. B.

The Pathophysiology of Coronary Artery Disease. G. Murray Annals of Surgery *126*: No. 4, 523-534, Oct. 1947.

The author postulates a revolutionary surgical treatment for coronary artery occlusion, as yet only in the experimental stage.

By tying off branches of the coronary in dogs, he has produced a sequence of pathological events similar to that occuring during coronary artery occlusion in humans. The resulting area of infarction if not placed so that the conducting system of the heart is involved and the heart thereby embarrassed soon becomes non-contractile and dilated. This acts as an expansion chamber into which blood is forced when the ventricle contracts. The effectiveness of contraction is partially lost because of this chamber, and the amount of blood delivered into the aorta less. This paradoxical systole is thought to be cause of many of the symptoms from which the coronary patient suffers.

The medical treatment of this condition can only be palliative. Surgical rescetion of the infarcted area to avoid formation of the expansion chambers has been carried out in dogs. Following operation there is an improvement in cardiac output, the blood pressure rises, and shock is alleviated. The survival rate is much higher than in a control group.

The treatment as applied to humans is as yet only in a speculative stage. In selected cases, immediate surgical operation might provide a better chance of survival from the effects of a very large infarct. A cure of the patient from an infarcted area of the myocardium would eliminate the dangers of acute rupture of the heart, or aneurysm and subsequent rupture.

H. M. B.

Cerebral Angiography: A Technique Using Dilute Diodrast. F. D. Ingraham and C. A. Cobb, Jr., J. Neurosurgery *4,* 422-434 (1947).

The authors present a method for visualizing the cerebral vascular networks using one or more injections of 10 cc. of a 35% solution of diodrast (perabrodil). This organic iodide is inert in the body, quickly excreted, and highly soluble. The common carotid or vetebral artery of either side is chosen and exposed. The injection is made with a special angiagram needle, 18-gauge curved type with rounded shoulder just above the bevel, attached to a syringe through a three-way stopcock.

The results from 25 operations are given. The patients ranged in age from 2

to 67 years. The diagnostic quality of the X-rays was satisfactory in all but two cases. There were positive findings in 9 cases and no undesriable effects in any.

H. A. P. M.

RECENT ACCESSIONS TO THE MEDICAL READING ROOM

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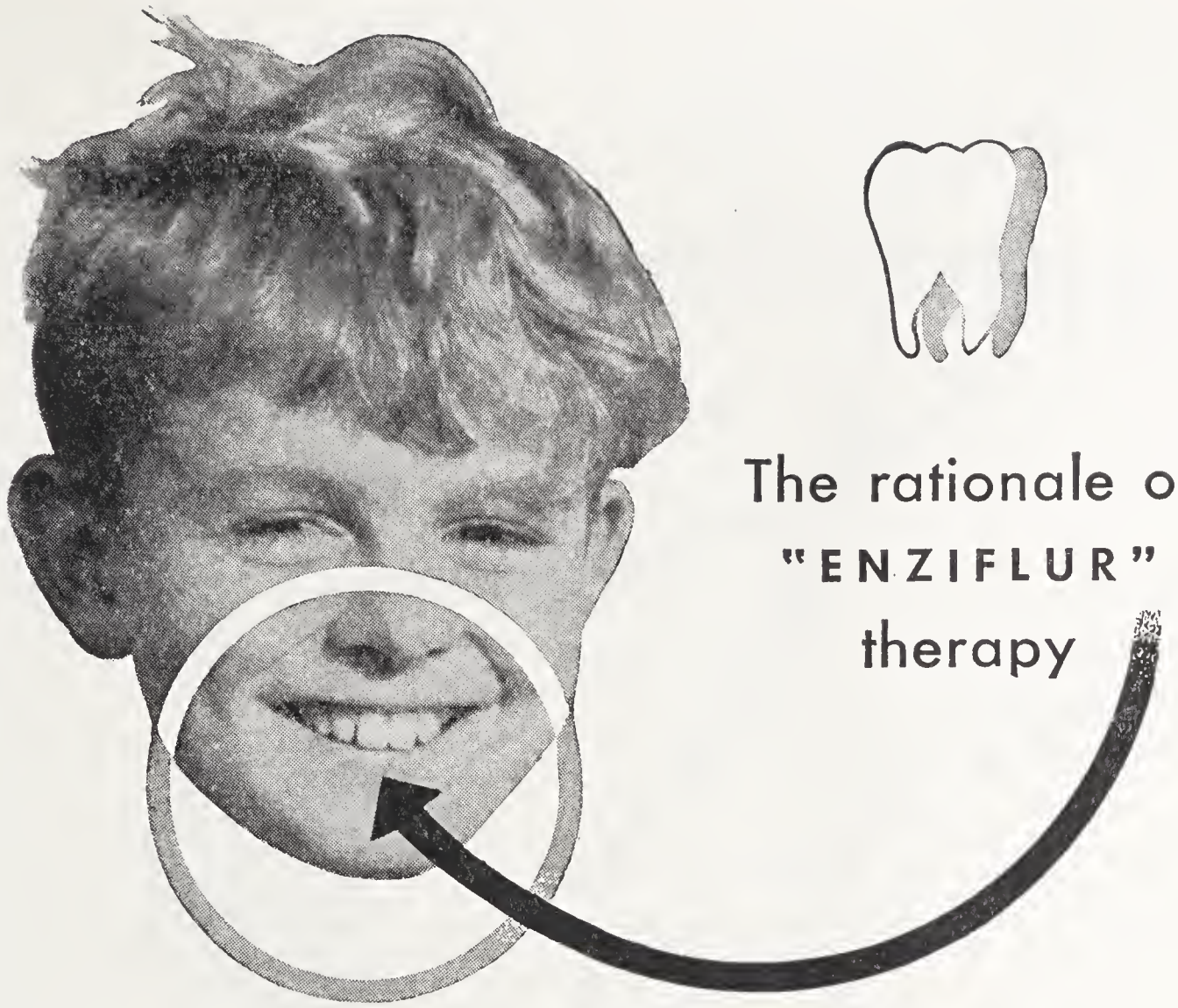

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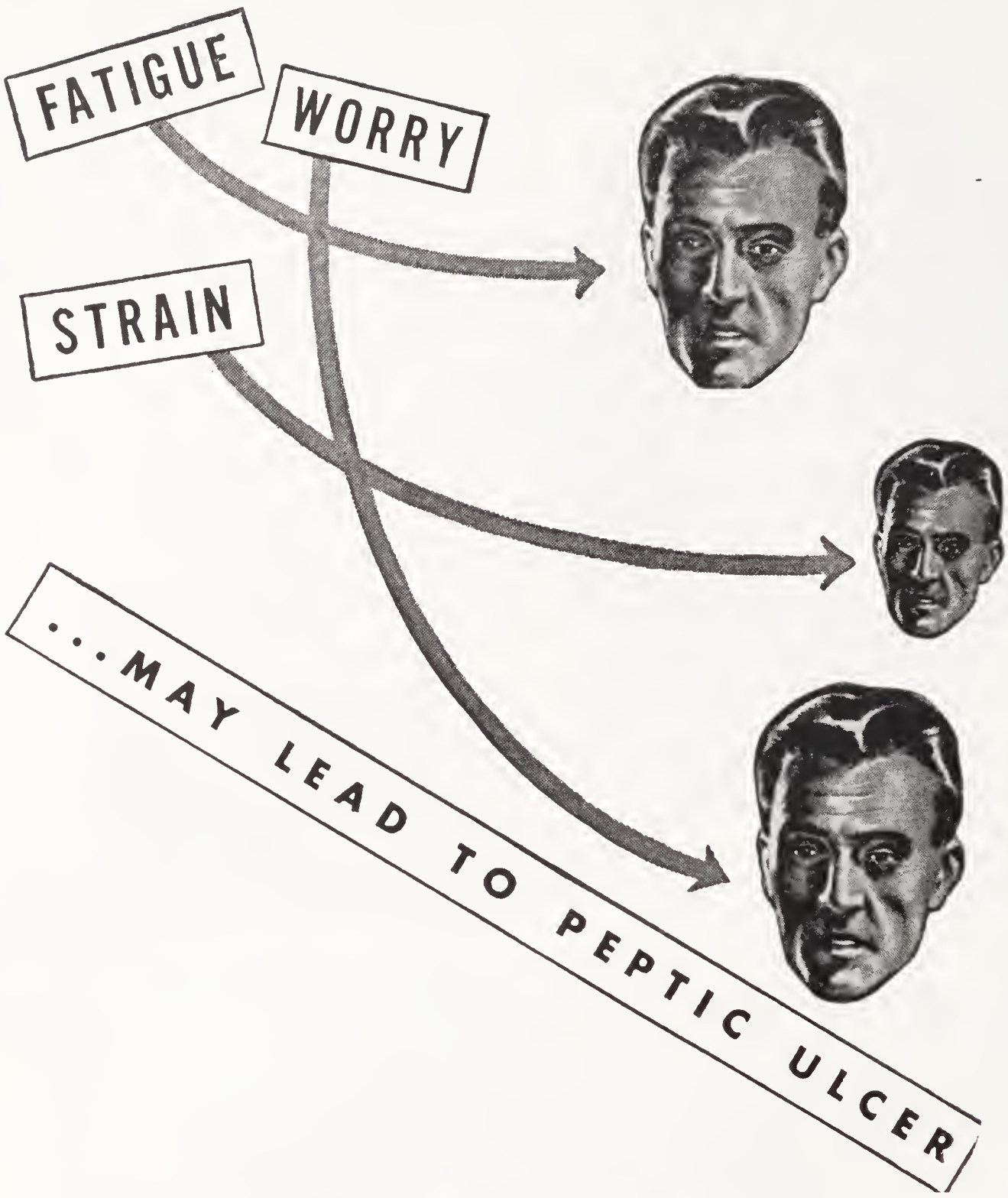


AMPHOJEL

ALUMINA GEL WYETH

IS THE MODERN TREATMENT

Amphojel is admirably suited to the management of peptic ulcer because it • precipitates pepsin • buffers gastric acid within normal limits • forms a protective coating over the mucosa.

Wyeth

Registered Trade Mark

JOHN WYETH & BROTHER (CANADA) LIMITED • WALKERVILLE, ONT.

SHOULD VITAMIN D BE GIVEN ONLY TO INFANTS?

VITAMIN D has been so successful in preventing rickets during infancy that there has been little emphasis on continuing its use after the second year.

But now a careful histologic study has been made which reveals a startlingly high incidence of rickets in children 2 to 14 years old. Follis, Jackson, Eliot, and Park* report that postmortem examination of 230 children of this age group showed the total prevalence of rickets to be 46.5%.

Rachitic changes were present as late as the fourteenth year, and the incidence was higher among children dying from acute disease than in those dying of chronic disease.

The authors conclude, "We doubt if slight degrees of rickets, such as we found in many of our children, interfere with health and development, but our studies as a whole afford reason to prolong administration of vitamin D to the age limit of our study, the fourteenth year, and especially indicate the necessity to suspect and to take the necessary measures to guard against rickets in sick children."

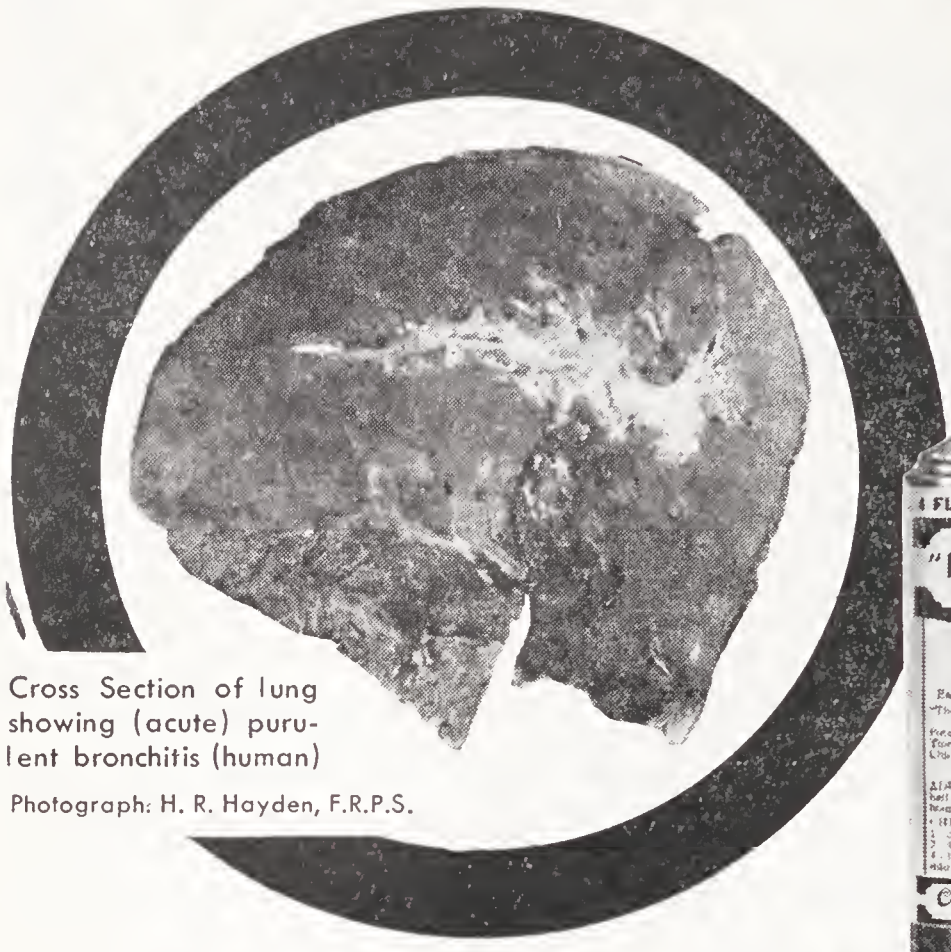
*R. H. Follis, D. Jackson, M. M. Eliot, and E. A. Park: Prevalence of rickets in children between two and fourteen years of age, Am. J. Dis. Child. 66:1-11, July 1943.

MEAD'S Oleum Percomorphum With Other Fish-Liver Oils and Viosterol is a potent source of vitamins A and D, which is well taken by older children because it can be given in small dosage or capsule form. This ease of administration favors continued year-round use, including periods of illness.
MEAD'S Oleum Percomorphum furnishes 60,000 vitamin A units and 8,500 vitamin D units per gram. Supplied in 10- and 50-cc. bottles and bottles of 50 and 250 capsules. Ethically marketed.

MEAD JOHNSON & CO. OF CANADA, Ltd., Belleville, Ont.

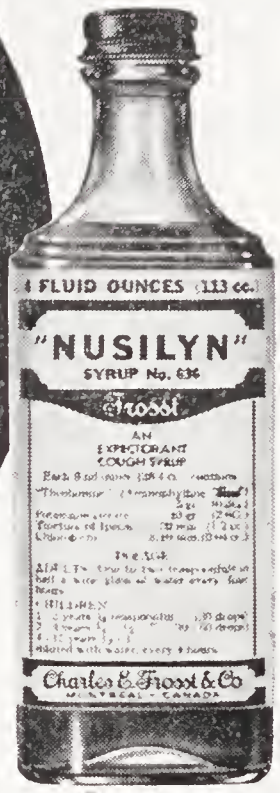
AN EXPECTORANT MIXTURE

for the Treatment of
ACUTE BRONCHITIS
CHRONIC BRONCHITIS
ASTHMATIC BRONCHITIS



Cross Section of lung showing (acute) purulent bronchitis (human)

Photograph: H. R. Hayden, F.R.P.S.



"NUSILYN"

During the exudative stage of acute respiratory infections, administration of Nusilyn tends to dilate bronchi and liquefy bronchial secretions and exudates. In this way expectoration is facilitated and the respiratory tract is cleared of obstructing secretions. Hacking and unproductive cough is avoided, patients are permitted rest and comfort, and healing is promoted.

Each Fluid Ounce Contains:

Theolamine (Aminophylline "Frosst")	5 gr.
Potassium citrate	40 gr.
Tincture of Ipecac	20 min.
Chloroform	8/10 min.
Simple syrup, flavoured	q. s.

"NUSILYN" with CODEINE

Nusilyn with Codeine (1 gr. per fluid ounce) is available for those cases in which part of the cough may be due to the pre-exudative stage.

DOSAGE

ADULTS: 1 to 2 teaspoonfuls.

CHILDREN: 1 to 2 years: ¼ teaspoonful (30 drops); 2 to 4 years: ⅓ teaspoonful (40 drops) to ½ teaspoonful (60 drops).

Well diluted with water and taken every four hours.

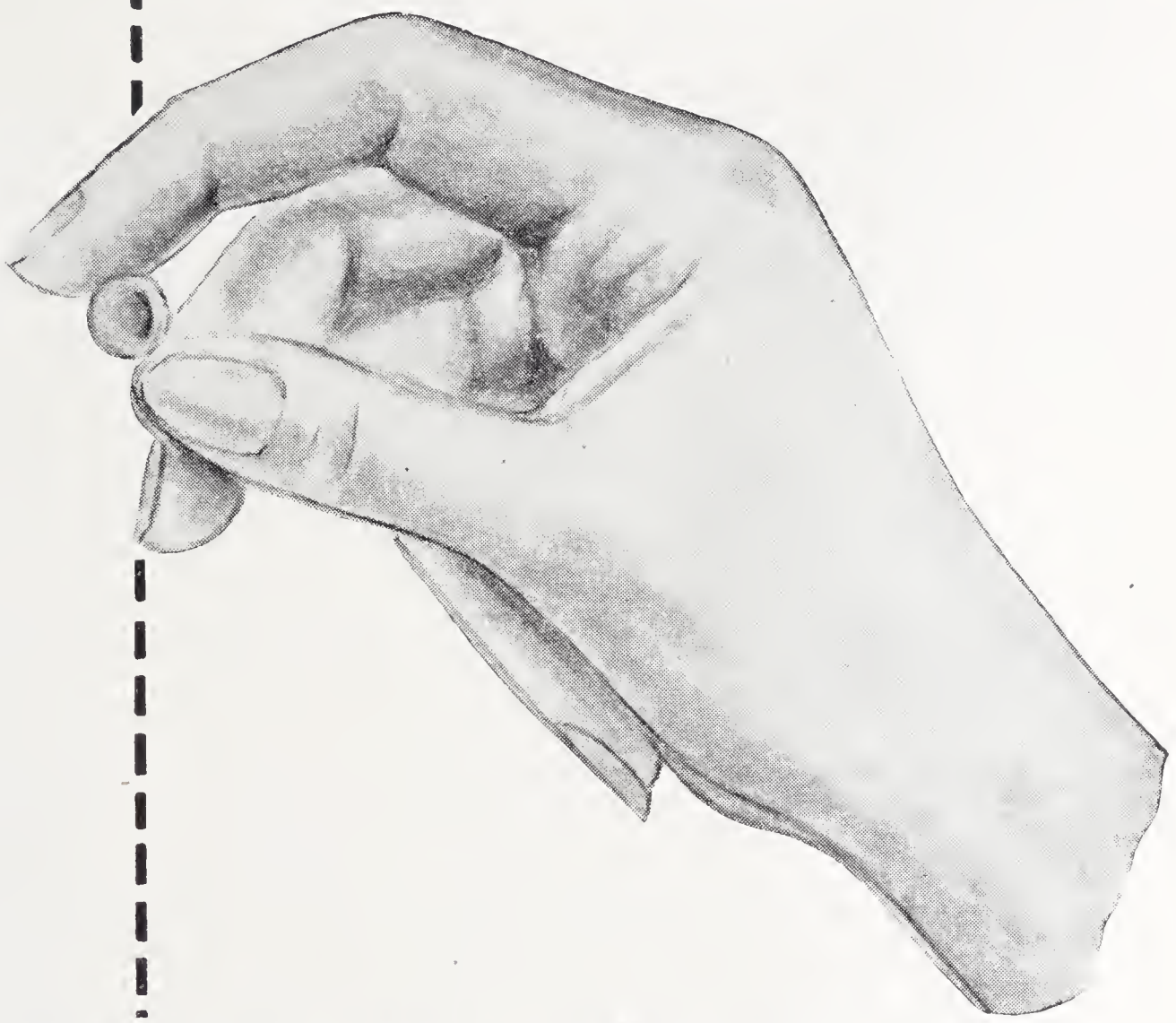
MODES OF ISSUE

Available in 4 fl. oz. 16 fl. oz., and ½ Imp. gal. bottles.

Charles E. Frosst & Co.
MONTREAL CANADA

Frosst
SINCE 1899
THE SYMBOL OF PROGRESS
IN PHARMACEUTICAL RESEARCH

ORALLY ACTIVE



HIGHLY POTENT • WELL TOLERATED

For menopausal serenity

CONESTRON TABLETS

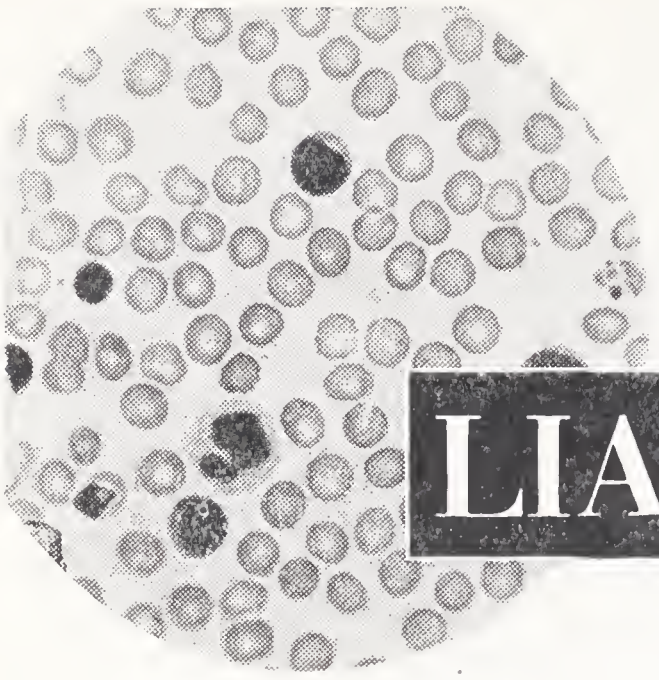
NATURAL CONJUGATED ESTROGENS

The physiologic transition incident to the climacteric may be gradual, or associated with disturbing and often incapacitating manifestations —headache, dizziness, hot flashes, and cutaneous, gastrointestinal, mental and other disorders. The usc of Conestron in these cases is a valuable aid to smoother, more rapid adjustment to the natural internal changes.

TWO STRENGTHS: Tablets of .625 mg. and 1.25 mg.

Wyeth

Registered Trade Mark



When it is difficult to categorize the anemia

LIAFON

SQUIBB

DESICCATED LIVER
FERROUS SULFATE
ASCORBIC ACID
FOLIC ACID

A new hematinic combination for the simultaneous administration of *four therapeutic essentials*

DESICCATED LIVER: whole liver with only the water removed. Provides nutritive elements of fresh liver, including the experimentally essential, clinically impressive secondary anti-anemia fractions.

FERROUS SULFATE EXSICCATED: one of the most readily utilized, tolerated and absorbed forms of iron. For *specific treatment* of iron deficiency anemias.

ASCORBIC ACID: often a prerequisite in anemias associated with C avitaminosis. Recent work also suggests it influences iron absorption and red cell maturation.

FOLIC ACID: bone-marrow stimulant factor of the B complex, *specific* for macrocytic anemias of malnutrition, pregnancy, pellagra, and sprue; also of value with parenteral liver therapy in Addisonian pernicious anemia.

Thus, when more than one form of anemia is present or suspected, and is difficult to categorize, *Liafon provides the essentials for therapy.*

EACH LIAFON CAPSULE CONTAINS:	DOSAGE EQUIVALENTS 3 capsules daily	DOSAGE EQUIVALENTS 6 capsules daily
Desiccated Liver ..... 0.5 Gm. (Approx. equivalent to 2 Gm. whole fresh liver)	*6 Gm. fresh liver	*12 Gm. fresh liver
Ferrous Sulfate Exsiccated . 2.0 gr. (Approx. equivalent to 2.85 gr. ferrous sulphate)	*8.5 gr. ferrous sulphate	*17 gr. ferrous sulfate
Ascorbic Acid ..... 50.0 mg.	150 mg.	300 mg.
Folic Acid ..... 1.67 mg.	5 mg.	10 mg.

*Approx. equivalent

Liafon is supplied in bottles of 100 and 1,000

For Literature write

E. R. SQUIBB & SONS CANADA LIMITED

36-48 CALEDONIA ROAD • TORONTO

31-8A

SQUIBB MANUFACTURING CHEMISTS TO THE MEDICAL PROFESSION SINCE 1858